RESEARCH

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ANAESTHETIC MANAGEMENT OF A POSTPARTUM PATIENT WITH SPONTANEOUS SUBDURAL HAEMATOMA DUE TO POSSIBLE HELLP SYNDROME: A CASE REPORT

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Abstract

HELLP (haemolysis, elevated liver enzymes, low platelet), a variant of preeclampsia, is a life-threatening pregnancy complication. It affects 0.5-0.9% of pregnancies globally. The mortality rate has been reported to be as high as 30%. We present a case of a 33-year old primigravidae delivered normally at 36 weeks of gestation, who sought treatment for preeclampsia. On third postpartum day, patient presented with drowsiness, headache and convulsion along with hemiparesis. Computed tomography scan revealed a large subacute subdural haematoma in the left fronto-temporo-parietal region, with oedema and midline deviation. Cerebral angiography showed a localised extravasation of the contrast media from cortical artery. Decompressive craniectomy for removal of subdural haematoma (SDH) was performed. She recovered after surgery with no residual neurological deficits. Thus, prompt recognition of intracranial haemorrhagic complications and timed neurosurgical intervention played key role in patient survival after such disastrous complication.

INTRODUCTION

The prevalence of preeclampsia, gestational hypertension and eclampsia were found to be 5.6 %, 1.5%, and 0.60 % respectively.[1,2,3] Preeclampsia is characterised by new onset hypertension (systolic blood pressure> 140 mm Hg and/or diastolic blood pressure> 90 mm Hg) and proteinuria (> 0.3g in 24 h) after 20 weeks of pregnancy. HELLP syndrome is an advanced stage of preeclampsia.

CASE REPORT

A 33year old primigravidae with preeclampsia, delivered normally full term, was referred on postpartum day 3, in our institute with complaint of headache, single episode of convulsion and right sided weakness. On examination, patient was afebrile, drowsy with Glasgow Coma Scale (GCS) E3V4M6 with right hemiparesis, left pupil mid dilated and sluggishly reactive, right pupil mildly constricted. There was no history or signs of head trauma. The cardiopulmonary evaluation was unremarkable.

Laboratory studies showed haemoglobin 8.2 g/dl, total leucocyte count 13700, platelet count 82000/mm3, random blood sugar 78 mg/dl, bleeding time 2.40 min, clotting time 4.45 min, prothrombin time 20 seconds, INR 1.8, total bilirubin- 1.5, aspartate transaminase (AST) 125 U/L, alanine transaminase (ALT) 142U/L, alkaline phosphatise (ALP) 190 U/L, lactate dehydrogenase (LDH) 1180 U/L, fibrinogen and D-dimer within normal limits.

CT scan of brain showed concavo-convex, extra-axial heterogeneously hypodense area of CT attenuation value of 31-38 HU, with few hyperdense area of CT attenuation value of 45-52 U, seen over fronto-parietal-temporal region on left side with maximum thickness of 1.5 cm.

Urgent intervention planned as per neurosurgical opinion. Patient positioned on the surgical table and multiparameter monitors (non-invasive blood pressure, pulse oximetry, urine output, electrocardiograph) were applied. Peripheral venous access secured with number 18G and fluid replacement started with heated Ringer's lactate solution. Scalp block infiltration for analgesia applied with 0.75% ropivacaine. Transfusion of 500 ml of fresh frozen plasma over 1hour and injection (inj.) vitamin K 10 mg and inj. tranexamic acid intravenous (i.v) were given.

During induction, inj. fentanyl 100mcg and inj. lignocaine 75mg i.v were given. After administration of inj. propofol 100mg and inj. rocuronium 60mg i.v, patient intubated with 7.5mm cuffed endotracheal

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tube via C-Mac guided laryngoscopy, bilateral air entry checked by auscultation. Anaesthetic depth was maintained by sevoflurane1-1.2% MAC and intermittent boluses of rocuronium. Mechanical ventilation presumed with volume cycle mode, tidal volume=8mL/kg and FiO2=50% in air. End tidal carbon dioxide (EtCO2) maintained in high normal range i.e. 25-30 mmHg. Left radial artery cannulated for continuous intraarterial blood pressure monitoring.

Decompressive craniectomy was done to evacuate the haematoma by left parietal and temporal burr hole. The surgical procedure was uneventful. Blood loss was 300ml. No blood transfusion was required. Serial ABG analysis showed no major acid-base imbalance. Postoperatively, with stable vitals, she was shifted to neurosurgery intensive care unit for elective mechanical ventilation and monitoring. Patient was extubated on second postoperative day after normalisation of haemodynamics and laboratory investigations.

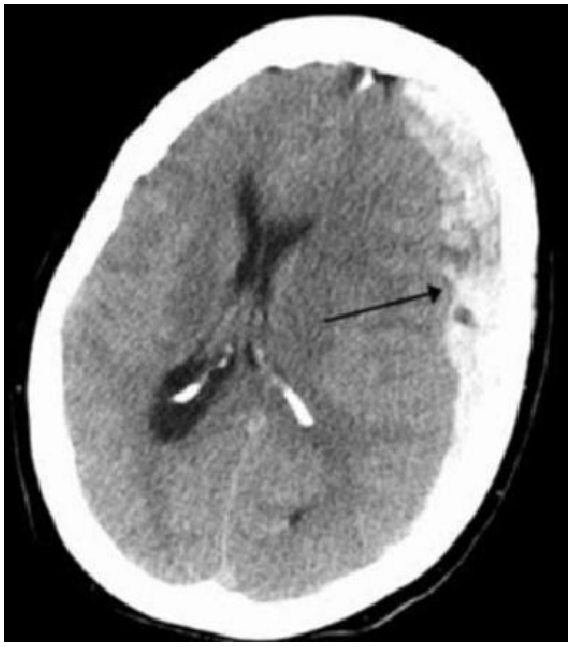

Figure. 1. Subdural Haematoma

DISCUSSION

In preeclampsia, abnormalities in the development of placental vasculature resulting in poor perfusion and hypoxia releases anti-angiogenic proteins soluble vascular endothelial growth factor receptor-1 and Fms-like tyrosine kinase, which bind to and inactivate placental and endothelial growth factors.[4] Continued vascular insufficiency of the placenta leads to platelet activation and fibrin deposition in the microvasculature, particularly in the liver.

HELLP syndrome affects 4-12% of those with preeclampsia.[1,4] Term coined by Dr. Louis Weinstein in 1982. Tennessee and Mississippi classifications divided HELLP syndrome as per [Table 1].

Table 1: HELLP

Mississippi classification	Tennessee classification
Class 1 • Platelets <50,000 • AST or ALT > 70 IU/L • LDH >600 IU/L	True or Complete • Platelets < 100,000 • AST > 70 IU/L • LDH >600 IU/L
Class 2 • Platelets = 50,000-100,000 • AST or ALT > 70 IU/L • LDH >600 IU/L	Partial or incomplete • Severe preeclampsia with any one of the following: ELLP, HEL, EL, LP
Class 3 • Platelets = 100,000-150,000 • AST or ALT >40 IU/L • LDH >600 IU/L	

ELLP, Absence of hemolysis; HEL, Absence of low platelets; EL, Elevated liver function; LP, Low platelets.

HELLP syndrome leads to complications such as pulmonary oedema, renal failure, disseminated intravascular coagulation (DIC), liver haematoma, postpartum bleeding, cerebral bleeding, and stroke.[5] Neurological disorders in pregnancy and postpartum are relatively common and can be classified into three categories,[6] 1) do not related to pregnancy status; 2) previous neurological disorders; 3) diseases whose incidence increases during pregnancy. This case report is included in the third category.

Brain-vascular disease is common in pregnancy due to fluid overload resulting in hypertension, high levels of oestrogen with known prothrombotic effect and endotheliopatia related to preeclampsia.

About 70% of HELLP cases develop before delivery, the majority between 27th and 37th gestational weeks; the remainder within 48 hours after delivery.[7] However, B. Cakmak et al,[8] showed evidence of late postpartum HELLP syndrome 60 hours after delivery in their study. K. Nakamura et al.[9] also suggested about late postpartum HELLP syndrome over 10 days after delivery.

Literatures related to spontaneous SDH in pregnancy associated with HELLP syndrome with concomitant parenchymal bleeding are limited. However, a study by M. Patabendige,[10] showed acute SDH in the postpartum period as a rare manifestation of possible HELLP syndrome with severe clinical manifestations upto 36th day postpartum.

Acute SDH with a thickness>10mm or midline shift> 5mm should be surgically evacuated Therefore, our patient is a suitable candidate for urgent surgical intervention.

The basic principles of neuroanaesthesia are provision of optimal operative conditions, maintenance of cerebral perfusion pressure and cerebral oxygenation. We ensured proper head positioning to reduce ICP and brain swelling by facilitating venous drainage. Multimodal analgesia provided with scalp block and opioids. Antiemetic drugs like dexamethasone and ondansetron were given. To prevent sympathoadrenal response, inj. lignocaine i.v given 60 seconds prior to intubation and C-Mac guided intubation done. Bite block, probes and thermometer insertion done with utmost care, preventing injuries because tongue and airway muscle bleeding may rapidly lead to airway obstruction postoperatively. Serial ABGs and blood glucose levels were monitored.

Anaesthetic agents like propofol, sevoflurane were chosen to maintain autoregulation and avoid increase in ICP. Nitrous oxide(N2O) avoided during

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anaesthesia course. Neuroprotective measures like maintaining cerebral oxygenation, hyperventilation to maintain eucapnia, drugs like mannitol (1gm/kg, iv), dexamethasone (8mg, i.v), seizure prophylaxis by phenytoin (100mg, i.v) were used.

CONCLUSION

Thus, to conclude HELLP syndrome leading to intracerebral haemorrhage is rare yet grave complication accounting for high maternal mortality. Prompt surgical decision alongwith anaesthesiologist playing pivotal role by providing adequate analgesia and sedation, maintaining stable haemodynamics, securing airway safely ensured favourable patient outcome.

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